EXHIBIT B

Occupational Medicine, Epidemiology and Prevention

October 28, 2021

Simmons, Hanly, Conroy
One Court Street
Alton, IL 62002
Attention: Melissa Schopfer, Esq.

Re: Brian Gref

Dear Ms. Schopfer:

I am writing to report the results of my evaluation of the materials listed below pertaining to Mr. Brian Gref. I have reviewed these materials in the context of my pre-existing knowledge, training, and experience in the field of occupational medicine. These materials are of the type I and other specialists in occupational medicine normally rely upon and are sufficient to form a reliable basis for my opinions contained within this report. All of the opinions stated in this report are given within a reasonable degree of medical certainty.

This report and the opinions stated in the report are based on the listed materials and my 30 years of training, education, and experience in the area of asbestos-related occupational medicine. Over the past 30 plus years, I have had the opportunity to evaluate and treat hundreds of patients with asbestos exposure, many of whom have asbestos related diseases.

Qualifications:

I am a physician licensed in the State of New York, specializing in the field of occupational and environmental disease. I have been a practicing physician since I graduated from medical school in 1988.

I attended the University of Chicago and received a Bachelor of Arts degree with Honors, with a major of History, Philosophy, and Social Studies of Science and Medicine. I then continued at the University of Chicago – Pritzker School of Medicine, where I obtained my medical degree in 1988. I was elected to the Alpha Omega Alpha Honor

Society, and was also awarded an American Medical Women's Association Award. Following medical school graduation, I was an intern and resident in Internal Medicine at Yale University – Yale New Haven Hospital from 1988 – 1991. Upon completion of my Internal Medicine Residency program, I completed a second residency at the Mount Sinai School of Medicine in Occupational Medicine, from 1991 – 1993. During my Occupational Medicine Residency Program, I obtained my Master of Science Degree in Community Medicine (equivalent degree to a Masters of Public Health) in 1993. I began to evaluate dozens of patients with asbestos exposure during my residency program at Mount Sinai. I am board certified in Occupational Medicine and in Internal Medicine. I have become recertified in Internal Medicine two times.

Following completion of my residency training in Occupational Medicine, I was awarded a Fellowship in Occupational Medicine from the Foundation for Occupational Health and Research. I continued at Mount Sinai, where I joined the faculty, and continued to evaluate patients with asbestos exposure. I became Vice Chair of the Department of Preventive Medicine in 2001. I was Director of the New York/New Jersey Education and Research Center from 2006 – 2010, and had been Director of the Residency Program in Occupational Medicine from 1998-2006. I was also the Director of the Mount Sinai World Trade Center Medical Monitoring and Treatment Program from 2006 – 2010, although my involvement with the World Trade Center medical programs started in 2001, when I began to evaluate patients with exposure to the World Trade Center disaster, and was initially Medical Core Director of the World Trade Center Worker and Volunteer Medical Screening Program (2002-2004), and Co-Director of the World Trade Center Medical Monitoring and Treatment Program (2004-2006). I have published over fifty articles in the peer-reviewed literature.

In 2010, I left the Mount Sinai School of Medicine to become the Founding Chair of the Department of Population Health at Northwell Health and Hofstra Northwell School of Medicine (formerly known as North Shore University Health System). The Department changed its name in 2014 to Occupational Medicine, Epidemiology and Prevention.

I have evaluated hundreds of patients with asbestos exposure in my career in occupational medicine, spanning over 30 years. I currently direct the Occupational and Environmental Medicine Center of Long Island, providing occupational health services to patients in the metropolitan New York area. Over the past year alone, I have supervised the examination of or directly examined nearly 700 patients with asbestos exposure, as we have greatly expanded our clinical services. Over the course of the past 30 years, I have evaluated dozens of patients with malignant mesothelioma and lung cancer due to asbestos exposure. I have kept abreast of the scientific and medical literature regarding the diagnosis and causation of mesothelioma. I have personally evaluated cases of mesothelioma where the exposure was brief, and have also seen cases of mesothelioma in individuals whose only exposure to asbestos was from family members who worked with asbestos and brought their asbestos contaminated clothes home.

My current rate for deposition and trial testimony is $750 per hour. My *curriculum vitae* is attached hereto as **Exhibit A**. A list of my former testimony is attached hereto as

Exhibit B. In addition to the documents referenced herein, I may also rely on the documents and literature referenced in my reference and reliance list for this case, attached hereto as **Exhibit C**.

Materials Reviewed:

I have had the opportunity to review the medical records of Mr. Gref and reviewed deposition transcripts from Mr. Gref and his parents. I was provided with the following information:

1. Dr. Zhang report
2. Advent Health medical records
3. Baptist Medical Center medical records
4. Cancer Specialists of North Florida medical records
5. Dr. Elizabeth Worsham medical records
6. Mayo Clinic medical records
7. VA Jacksonville medical records
8. Dr. Mark Krekler reports
9. Social Security records
10. Plaintiff's Answers to Interrogatories
11. Deposition of Brian Gref
12. Deposition of Roger Gref
13. Deposition of Jeneane Bennett
14. Article-Asbestos Found in Ten Powders, dated 3/10/76
15. Memo from Environmental Science Laboratory dated 3/22/76

Mr. Gref's Medical and Exposure History:

Clinical History: Mr. Gref is a 38 year old who presented to the Emergency Department (ED) on August 17, 2017 with right flank pain. A CT scan showed a right ureteral calculus. He returned to the ED on December 4, 2017 with left upper quadrant abdominal pain, fever, chills, nausea and vomiting for four days. A CT scan of the abdomen and pelvis on December 4, 2017 showed moderate induration of the omentum in the left upper quadrant and a small amount of free fluid in the pelvis. These findings were new compared with the CT scan in August 2017. There was a concern for ischemia to the omentum, and suspected omental torsion. Mr. Gref underwent surgery on December 6, 2017 and an exploratory laparotomy with omenectomy was done, with an area of congestion noted in the omentum. The pathology showing acute inflammation, reactive mesothelial hyperplasia and vascular congestion. There was no evidence of malignancy. He was discharged home on December 7, 2017. Mr. Gref had another episode of abdominal pain in late October 2018. A CT scan of the abdomen and pelvis showed a very small area of omentum to the right of the midline just above the umbilicus that showed hazy edema. There was a hazy density in the left upper quadrant mesentery near the spleen. A loop of small bowel was mildly dilated with slight thickening as well. There was "a swirling of the deep mesentery without vascular occlusion." The radiologist's impression was mesenteritis. He was admitted to the hospital and treated with intravenous fluids, antibiotics, and pain medication. He underwent an

endoscopy on October 30th, 2018 that was normal. His pain resolved and he was discharged home.

Mr. Gref developed abdominal pain again on December 17, 2018, and went to the ED, where a CT angiogram showed no evidence of an acute vascular abnormality. There was circumferential mucosal thickening of the small bowel loops within the left mid abdomen and left upper quadrant, most suggestive of small bowel enteritis. There was redemonstration of inflammatory stranding/edema throughout the mesenteric fat especially within the left upper quadrant. There was significant interval progression compared to the previous examination, and the radiologist noted that while the findings were non-specific, they did raise the possibility of peritoneal carcinomatosis. He was admitted to the hospital and given fluids and antibiotics. A significant fluid collection was noted in his abdomen, and a paracentesis was done on December 19, 2018. Around 1700 cubic centimeters of fluid was removed and the cytology was negative for malignant cells. There were some reactive mesothelial cells, along with neutrophils and lymphocytes. A video capsule endoscopy showed slow gastric transit, gastric ulcerations and gastritis and a normal small bowel otherwise with delayed transit. The endoscopy biopsies showed findings suggestive of peptic duodenitis and gastritis. No abnormalities were noted in the colon. He was discharged home on December 23, 2018.

Mr. Gref's abdominal pain returned in June/July 2019 and he developed nausea and difficulty taking a deep breath due to left upper quadrant pain. The pain gradually worsened over three to four weeks and he returned to the ED for evaluation on July 20, 2019. He had a CT scan of the abdomen that showed a moderate amount of ascites throughout the abdomen. Small bowel thickening was seen within several loops of small bowel within the abdomen. He was placed on antibiotics for presumed mesenteritis. An endoscopy was done that showed gastritis, and the gastric antrum showed no significant findings. He had a paracentesis done with 1600 cubic centimeters of fluid removed, and the fluid cytology showed acute inflammation and mesothelial cells. No malignant cells were seen. He was discharged home on July 22, 2019. A repeat CT scan was done on August 18, 2019. There was a moderate amount of intra-abdominal ascites. Mild splenomegaly was seen. There were a few mildly enlarged epicardial lymph nodes. Mr. Gref was admitted to the hospital with five days of abdominal pain and vomiting. Dr. Bradford Joseph, a gastroenterologist, saw Mr. Gref in the hospital. He recommended an MRI of the abdomen and Doppler ultrasound of the liver. If the tests were abnormal, he recommended a surgical evaluation. A repeat paracentesis was done with 1,400 cubic centimeters of fluid removed. Dr. Benjamin Piperno, a surgeon, saw Mr. Gref in consultation. He did not feel he required surgery at that time. An MRI of the abdomen was done on August 20, 2019. There was a moderate volume of ascites of uncertain etiology. Borderline enlarged epicardiac lymph nodes were again seen. There was a tiny splenic lesion, likely benign, and mild splenomegaly. A less than one centimeter pulmonary nodule was seen at the right lung base. An abdominal ultrasound showed mild splenomegaly and small volume ascites. Normal blood flow in the portal vein was seen on Doppler. A paracentesis was done and the fluid showed reactive mesothelial cells and acute inflammation. No malignant cells were seen. He was discharged home on August 25, 2019, and a recommendation was made to seek care at Mayo Clinic for a second opinion.

Mr. Gref was admitted to the Mayo Clinic in Florida on Novembe 24, 2019 with worsening abdominal pain and distention along with nausea. He also had non-tender cervical lymphadenopathy. A CT scan on November 24, 2019 showed a large volume of ascites with heterogenous stranding and nodularity of the omentum, omental caking with a soft tissue mass anterior to the level of the umbilicus. Mr. Gref had a CT-guided paracentesis and biopsy. Around 2.15 liters of fluid was removed from the abdomen. The fluid showed neutrophils and he was treated with antibiotics and given albumin to treat spontaneous bacterial peritonitis. The fluid cytology and the biopsy pathology showed malignant mesothelioma, epithelial cell type. Dr. Zhang reviewed the pathology and noted that the histological features and immunohistochemical staining pattern supported the diagnosis of malignant mesothelioma, epithelioid cell type. Mr. Gref was switched to oral antibiotics and plans were made for an outpatient chest CT, PET/CT and consultation, along with surgical consultation regarding possible HIPEC. He was discharged home on November 28, 2019. Mr. Gref returned on November 29, 2019 with a panic attack. He was treated with anxiolytics.

Dr. Thomas Davis, a medical oncologist, saw Mr. Gref on December 15, 2019. A PET scan was done on December 17, 2019 and showed hypermetabolic omental soft tissue infiltration throughout the upper abdomen consistent with known mesothelioma with a mass in the right upper quadrant measuring 3.2 x 2.5 centimeters. There was moderate ascites with low-level FDG. There was no abnormal uptake within the neck or chest. Dr. Marcelo DaSilva, a surgical oncologist, saw Mr. Gref on January 8, 2020. He discussed treatment options with Mr. Gref including cytoreductive surgery. Mr. Gref went to the ED at Mayo Clinic on January 21, 2020 with two weeks of abdominal pain and nausea. A bedside ultrasound showed no ascites. He was given pain medication. Dr. DaSilva recommended induction chemotherapy with Cisplatin and Pemetrexed, and Dr. Davis planned to administer the chemotherapy and rescan after two cycles. Mr. Gref went to the ED on February 8, 2020 with chest pain, shortness of breath and generalized weakness. He had started chemotherapy on February 4, 2020. A CT angiogram of the chest showed no evidence of a pulmonary embolism. There was no acute abnormality noted in the chest. His chest pain was relieved by nitroglycerin. He was admitted for monitoring to see if he had a cardiac event. He continued to have abdominal pain and nausea. A stress echocardiogram showed normal clinical response to exercise stress with no chest pain during the procedure. There was a normal electrocardiographic response during exercise stress and perfusion imaging was normal. The overall left ventricular systolic function was normal with no wall motion abnormalities. He was discharged home on February 10, 2020.

Dr. Davis saw Mr. Gref on February 18th, 2020. He had some nausea, headache and diarrhea, and was getting a bit dehydrated. He had less abdominal distension. Dr. Davis arranged for hydration and planned a second cycle for the following week. Mr. Gref saw Dr. Davis on March 10th. He had daily nausea and a poor appetite with occasional diarrhea. He received intravenous fluids and anti-emetics. He received his third cycle on March 17th, 2020. Dr. Davis saw Mr. Gref on March 31st. His nausea and appetite were improved but he still had nausea and bloating for a week after treatment. A PET scan was done on March 31, 2020. There was an interval decrease in the volume and FDG intensity within the

mesothelioma in the omental fat, consistent with a response to interval therapy. There were persistent areas of fine omental nodularity in the left upper quadrant with low-level FDG activity. Persistent but improved intra-abdominal ascites was noted. Dr. Davis saw Mr. Gref on April 7th. Mr. Gref was doing better on treatment and his pain had decreased. He was using Varubi and Protonix with relief of his symptoms. Dr. DaSilva saw Mr. Gref via telehealth on April 22, 2020 and discussed the plan for surgery. Dr. DaSilva planned a radical peritonectomy with possible cholecystectomy, splenectomy, bowel resection and omentectomy with HIPEC. Dr. Davis saw Mr. Gref on April 28, 2020. He planned to see him in June following his surgery.

Mr. Gref underwent cytoreductive surgery with HIPEC on May 20, 2020. Dr. DaSilva performed a radical omentectomy and administered intraoperative heated Cisplatin. [full records of the surgery and hospitalization were not available for my review]. The pathology showed multiple miscroscopic foci of malignant mesothelioma in the peritoneum soft tissue. There was extensive malignant mesothelioma involving an area up to 30.3 centimeters and excision of the mesentery showed malignant mesothelioma. Dr. DaSilva saw Mr. Gref on July 8, 2020. He was able to walk around ½ of a mile a day and had very mild incisional pain. A PET scan on July 28, 2020 showed no PET evidence of active malignancy. There was interval resolution of the previously seen fine nodularity that was infiltrating the omental fat in the left upper quadrant. No intraperitoneal ascites was seen and no focal abnormality was seen in the neck or chest. Dr. Davis saw Mr. Gref on July 29, 2020. Dr. Davis noted that there was no evidence of disease on his PET scan. He started him on lisinopril for an elevated blood pressure. Mr. Gref returned to Dr. Davis on March 10, 2021. Mr. Gref continued to require pain medication and Dr. Davis noted he had no evidence of disease on his recent scan [not available for my review]. He planned to see Mr. Gref in 12 months for follow-up with a repeat scan.

No other records were available for my review.

Past Medical History: Mr. Gref has a history of kidney stones, chronic sinusitis and sinus surgery, migraines, low back pain, bipolar disease, alcohol use disorder, depression and anxiety.

Cigarette Smoking History: Mr. Gref smoked cigarettes regularly from 2004 – 2019. He smoked between several cigarettes a day to up to one-half pack of cigarettes a day, although there is notation that he smoked around one pack a day.

Environmental and Occupational History: When Mr. Gref was young, his parents served in the US Navy. He recalled his mother worked in an office and as a detailer and his father was a drug and alcohol counselor and worked on aircraft, working on ejection seats and doing supervisory work. His mother had ship duty, but she did not go into the mechanical areas, and performed administrative duties onboard. His father was unaware of any asbestos exposure during his service. His parents separated when he was young and he and his mother moved to Virginia from Florida when he was around 9. Mr. Gref worked as a security guard from 2001 – 2004. He worked at a front desk for an office building for two years from 2003 – 2005. He worked as a service technician for North Florida Lube

from 2005 – 2007. He worked as a call center operator from 2007 – 2013, and served in the US Army from 2014 – 2015. He worked as a website builder from 2015 – 2019, and has been a blood technician since 2019.

Mr. Gref recalled that his parents bathed him when he was young and dried him off and applied powder to his body. He recalled that a lot of powders were in the house, particularly Old Spice and Clubman, which were used a lot. He also remembered English Leather, Johnson & Johnson, Shower to Shower, and Mennen. He stated that the powder was applied "in healthy doses, dust getting all up in the air." His parents either applied the powder directly onto him from the bottle or put the powder on their hand and applied it over his body and rubbed it in. Mr. Gref stated that his family no longer used Mennen after 1993 or 1994. When Mr. Gref moved with his mom to Virginia in 1991, he stopped using powder daily, and used it weekly until he was 12 years old. He used the same brands of powder in Virginia. When he was around 12 he began using powder daily. He applied it to his groin region, under his arms, behind his knees, and to his neck. Mr. Gref used a container of powder every two to three weeks. Mr. Gref graduated from high school in 2001 and continued to use the powders, including Johnson and Johnson, Shower to Shower, Clubman and Old Spice, on a daily basis. After he moved to Florida in 2004 Mr. Gref continued to use powders by English Leather, Clubman, Johnson and Johnson and Shower to Shower. He used these powders until he was diagnosed with mesothelioma in 2019. Mr. Gref stated that he used the various powders interchangeably. He estimated he would go through around 6 containers of Clubman talcum powder a year. He had to clean up the bathroom after he used the powder, around two to three times a month. He estimated over his lifetime he used dozens of bottles of Clubman talc over the years he used powder. He used several dozen bottles of English Leather talc, and ten to twelve bottles of Old Spice (during the period of 1994-1999/2000)]. He also mentioned using Imperial Talc.

Mr. Gref's father, Roger Gref, noted that he used Johnson's Baby Powder or Mennen when he changed his son's diapers. He also applied talcum powder after Mr. Gref was bathed. His mother, Karen Nappi recalled using Mennen, Clubman, Shower to Shower, Old Spice, Johnson and Johnson's Baby Powder, and English Leather on her son. When she and Mr. Roger Gref were stationed in Cuba, her father would send packages that included talcum powder for her to use on Mr. Gref, who was age 3 months to 3 years. She used various powders when she was changing his diaper, and then after his bath. Ms. Nappi used powder on Mr. Gref until he was around 7-8 years old, when Mr. Gref began bathing independently. In Cuba she used three to four containers once a month for 30-36 months. Over the years from 1982-1987, Ms. Nappi estimated that she used 5-7 bottles each of the various talcum powders on her son. She estimated that she bought around 5-7 bottles of Mennen and the other powders when Brian was 7-11 years old. He used more after he was around 11. Similarly, she bought additional bottles of the various talcum powders for him more frequently after he reached puberty, around 11 or 12, and he continued to use a variety of powders.

Conclusion: Mr. Gref has malignant mesothelioma of the peritoneum as a result of his cumulative exposure to asbestos. His parents noted that they used talcum powders on Mr. Gref from birth, including Mennen, Clubman, Shower to Shower, Old Spice, Johnson's

Baby Powder and English Leather. Mr. Gref continued to use talcum powders until 2019, for a total of 36 years. Mr. Gref has undergone chemotherapy and cytoreductive surgery.

The methodology and basis for my opinions follows standard methods of the medical and scientific community and have been described in the peer reviewed literature including by Bradford-Hill, Lemen, and Welch. Asbestos is the most well known cause of mesothelioma, and the causation of mesothelioma has been established by the quantitative history of exposure to asbestos. Thousands of individuals, from myriad professions and exposure situations have developed mesothelioma as a result of either direct or indirect exposure to asbestos. The reliance on the history of exposure to asbestos was used by seminal studies by Newhouse, Wagner and Selikoff in the 1960s, who attributed mesothelioma to asbestos exposure based solely on the history of exposure. The increased risks for mesothelioma exist for individuals who both directly handled asbestos products and for those who worked adjacent to or were in the vicinity of others who were using asbestos products, which is known as "bystander" exposure.

Asbestos and Malignant Mesothelioma General Opinions: Occupational Medicine is the field of medicine that deals with exposures to substances, toxins, conditions and agents in the workplace that are associated with increased risks of diseases. It exists as a subspecialty of Preventive Medicine that deals with identifying ways to prevent people from becoming ill. This includes identifying the sources, agents or catalysts that increase the likelihood of someone developing a disease, illness, or detrimental condition, and educating people on how to eliminate, avoid, and/or mitigate those risks. To put it simply, Occupational Medicine and Preventive Medicine involves searching for and identifying causes of diseases. This knowledge is important for those who are already ill: elimination of the catalysts can eliminate or mitigate the illness. It is also important from a public health point of view: to a large extent, the higher purpose of Occupational Medicine and Preventive Medicine is to educate and warn the public on how to eliminate, avoid, or mitigate the risks of diseases at the workplace, and to provide guidance to governments and businesses on appropriate regulations and standards concerning workplace health and safety.

One of the essential tasks of a physician of Occupational Medicine, when dealing with an individual patient, is the taking of a proper occupational history. Standard medical histories usually involve the patient explaining their reason for seeking medical attention; a listing of current symptoms, conditions, allergies, medications and other relevant medical problems; and providing some family and social history. Occasionally, a standard medical history may-but doesn't always-include identifying the patient's occupation.

A full occupational history, on the other hand, will go into details of a patient's entire work history, including details concerning their tasks and duties and their working conditions and environment. The history will also routinely make inquiries into the patient's home or hobbies. It would also reveal what kinds of substances or agents the patient was exposed to in his or her working environment that might have occurred decades earlier. It remains the standard tool for determining exposure and has not been supplanted by quantitative measurements, which are rarely obtained, and would not, unless continuously performed on an individual (which is not feasible), fully address all exposures

an individual might have had. At times, it is not possible to directly obtain an occupational history from an individual, and information concerning work and environmental experiences contained in deposition transcripts by plaintiffs, co-workers and family members can provide detailed information of that type that can be elicited from an occupational physician-obtained history.

The hallmark of occupational medicine is to connect an exposure to a hazardous substance to a disease, and identify whether there is a causal relationship. This is a critical differentiation in the field of occupational medicine; not only do we treat patients for disease, but we emphasize what hazardous substance might be causing the disease. In occupational medicine training, there are core areas of training, including epidemiology, biostatistics, toxicology, and industrial hygiene.

Asbestos and Disease: Asbestos is a naturally occurring mineral that has been used commercially for a variety of purposes for over 100 years. Asbestos is mined in the form of microscopic fibers released from the surrounding earth. Asbestos was extremely useful from an industrial perspective: it is highly resistant to heat and therefore serves as an excellent insulator and friction surface. It is also very durable, and as a fiber it can be molded into shapes and products that serve a variety of functions. However, asbestos is also highly toxic and carcinogenic when the fibers are inhaled or ingested.

While there are many "fiber types" of asbestos, as well as different sizes of the fibers, there exists consensus among scientists that exposure to *any* asbestos fiber type or size increases the likelihood of lung cancer, mesothelioma, as well as nonmalignant lung and pleural disorders. Asbestos fibers are generally divided into two categories: amphiboles and serpentine (or chrysotile). There are several varieties of amphiboles, including both commercial and non-commercial types. The three major asbestos types used in industry have been chrysotile, amosite and crocidolite. Of these three fiber types, over 95% of all asbestos used in the United States has been chrysotile. Much of the chrysotile asbestos that was used in the US was mined in Canada, where there was contamination with small amounts of tremolite, another type of amphibole asbestos. The mainstream scientific community has also long recognized, and continues to recognize today, that there is no "safe" level of exposure to asbestos regardless of fiber type or size. This position is shared by numerous United States government agencies, including the Occupational Safety and Health Administration ("OSHA", which has regulatory authority over workplaces), the Environmental Protection Agency ("EPA" which has regulatory authority over non-occupational settings), the National Institute for Occupational Safety and Health ("NIOSH", which is responsible for conducting research and making recommendations for the prevention of work-related injuries and illnesses), the World Trade Organization ("WTO"), and the national academies of science of every major industrialized nation. The World Health Organization recently reviewed the existing literature and concluded (in 2014) that all fiber types are capable of causing asbestos related disease, including mesothelioma, and reiterated the statement that there is no safe level for exposure to asbestos.

Due to the ubiquitous use of asbestos and its presence in naturally occurring formations, there is asbestos in the ambient air in the United States, albeit at minute levels. The ambient air concentration or "background level" has been reported to be approximately 0.00001 f/cc. These levels are thousands of times less than the current OSHA permissible exposure level of 0.1 f/cc. While it is theoretically possible to develop mesothelioma from ambient air concentrations, it has not been proven to occur at levels at or below ambient air concentrations. Given that there is no truly "unexposed" population, it would be impossible to reasonably perform such a study to determine if this were the case.

State of the Art (Brief):

In 1898, Montague Murray described interstitial fibrosis in an individual exposed to asbestos. Pancoast described radiographic changes of interstitial fibrosis in asbestos workers in 1917. Cooke described two cases of asbestosis in the 1920s, and actually used the term "asbestosis" to describe the interstitial fibrosis among asbestos workers, and also noted pleural plaques (fibrosis) in these workers.

In 1930, Merewether and Price, in their *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry,* noted that inhaling dust containing asbestos fibers could lead to disabling and fatal lung disease. They studied asbestos workers in the textile mills in Great Britain, and noted that asbestosis could occur in large numbers of exposed individuals. Moreover, they found that the textile workers with the highest exposures had more asbestosis than workers in areas where asbestos exposure was lower. Merewether and Price noted that asbestos was a potential hazard to health in any industry where dry asbestos products were abraded or otherwise manipulated to generate dust, such as thermal insulating. They recommended warning, education and training of all those individuals who were exposed to asbestos.

Lynch and Smith noted a case of lung cancer in an asbestos worker from South Carolina in 1935. Textbooks in the 1930s, such as A.J. Lanza's textbook on dust disease, included asbestosis as a disease of concern. In 1943, the first case of mesothelioma was associated with asbestos exposure and was published by Wedler in Germany. Also in 1943, Hueper from the United States Public Health Service stated that he believed asbestos caused lung cancer. He published an editorial stating this association in the Journal of the American Medical Association in 1949.

In 1955, Doll published a seminal article that described the increased risk of lung cancer among asbestos exposed workers. By the time of Doll's epidemiology study, there had been over 60 cases of asbestos-related lung cancer published in the literature. In 1960, Wagner et.al. published a study of 33 cases of malignant mesothelioma among individuals who were exposed to asbestos in and around the crocidolite mines in South Africa. Not only were miners developing disease, but family members, individuals on the wagon routes in which the asbestos was carried and people who had played with mine tailings as children developed mesothelioma. In the early 1960s, numerous studies in several countries, under different exposure scenarios, were published that showed mesothelioma in association with

asbestos exposure. In fact, by the end of 1964, over 700 scientific articles had been published that showed the adverse health effects of asbestos.

Asbestos Related Disease:

The Development of Diseases: When asbestos is inhaled, some proportion of the fibers can be deposited upon any component of the respiratory tract, including the nose, pharynx, conducting airways and the alveolar or gas exchanging regions of the lung. Fibers that land initially on the airways and above are cleared rapidly from the lung. The primary defense mechanism that mediated this clearance is known as the mucociliary escalator. The escalator is comprised of collated and mucous producing epithelial cells that propel inhaled fibers up to the mouth where they can be swallowed or expectorated. These epithelial lining cells are the "target cells" for cancers. Fibers that evade the mucociliary escalator can penetrate into the lower airways and lung tissue, where they can be transported through the body. Amphibole fibers tend to clear from the lung less rapidly than chrysotile fibers. Asbestos is cleared through the pulmonary lymphatics to lymph nodes and to the pleura, the target organ for pleural mesothelioma. Of the different fiber types, Suzuki, Sebastien and LeBouffant have all shown that chrysotile fibers preferentially translocate to the pleural space.

Asbestosis: The fibers that are inhaled and deposited past the escalator can cause asbestosis. These fibers deposit initially on the Type 1 and Type 2 alveolar epithelial cells. On the epithelial surfaces, some asbestos fibers activate the 5th complement which attracts inflammatory cells, including foreign particles, like asbestos, from the lung. About 20% of the fibers deposited on the alveolar surfaces are enveloped by the Type 1 cells and are translocated to the underlying connective tissue (interstitial) compartment. There, the fibers can interact with interstitial fibroblasts, myofibroblasts and macrophages. Fibroblasts and myofibroblasts are the target cells for asbestos because these are the cells that synthesize and release the scar tissue matrix. (See Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues.,*19 Am J. Indus, Med. 701 (1990); Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues*., *19* Am J. Indus, Med. 701 (1991)). They produce scar tissue when the epithelial cells are injured and when the macrophages are activated. Alveolar cells and macrophages release a number of protein growth factors that stimulate the fibroblasts to multiply and produce scar tissue and the fibroblasts and myofibroblasts also synthesize a similar array of factors that induce their own cell growth and matrix production that we recognize as asbestosis. Like *all* of the asbestos-related diseases, asbestosis is dose dependent. An individual typically needs long-term occupational exposure to develop clinical asbestosis.

The scarring process described above begins as soon as inhaled fibers are deposited on the alveolar surfaces, and microscopic asbestosis is ongoing in the lungs of afflicted individuals for many years before any clinical signs or symptoms are presented. The initial physiological symptom of asbestosis is shortness of breath. This is caused by the scar tissue which replaces normal elastic connective tissue, this producing a stiff lung that restricts the individual from taking a deep breath. Shortness of breath also results when scar tissue

thickens the alveolar-capillary membrane, the barrier across which oxygen and carbon dioxide gases are exchanged.

Pleural Plaques and Fibrosis: This is scar tissue formation in an identical manner to that described above, under asbestosis. The difference is that there is little direct deposition of asbestos fibers in the pleura. While some fibers can be inhaled through the alveolar ducts and reach the pleura directly, most fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to the pleura do so by way of pulmonary lymphatic flow. The inhaled fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to lymphatic fluids which flow through these regions on the way to the pleura. The lymphatic flow carries fibers to the pleura where they interact with the sub-mesothelial fibroblasts that produce a scar tissue matrix, as described above. If the scarring is in a circumscribed pattern, the scarring is called "plaque". Investigators have shown that this injury can result in a restrictive lung disease in some individuals.

Lung Cancer: These tumors caused by asbestos typically arise in cigarette smokers, although some epidemiologic studies on asbestos-exposed non-smokers show an increased risk of developing the disease. When an individual is exposed to the cancer-causing agents (carcinogens) of both cigarettes and asbestos, the risk of getting lung cancer is increased well beyond the risk presented by exposure to either agent alone or by simply adding the risks of the two carcinogens. Epidemiologists multiply the risks of the two carcinogens since there is a clear synergy in the way asbestos and cigarette smoke combine to cause lung cancer.

Cancer is the loss of control of cell growth. Every cell in the bodies of humans and animals is under strict genetic control of the rate at which a given cell replaces itself by dividing. Cancer is caused when the specific genes that control cell division and other aspects of the cell cycle develop errors or mutations. Carcinogens induce such errors, and complete carcinogens can produce the errors with no other agent required. Cigarette smoke has a number of complete carcinogens, and all of the asbestos varieties have been shown to act as complete carcinogens. Thus, as the airway epithelial cells of the mucociliary escalator are assaulted daily by cigarette smoke and asbestos fibers, a number of cells are injured, and many exhibit genetic errors through the lifespan of the individual. In those who are susceptible to developing a cancer, one of those injured cells accumulates a sufficient number of genetic errors in genes that control cell growth to finally, after decades of exposure, lose the normal growth pattern and grow into a malignant tumor. (See Frost G, Darton A, Harding AH. *The effect of smoking on the risk of lung cancer mortality for asbestos workers in Great Britain (1971-2005)* Ann Occup Hyg 55:239-24 (2011)).

Mesothelioma: This cancer occurs when mesothelial cells of the pleural or peritoneal surfaces develops a sufficient number of genetic errors in a set of genes that control cell growth, as described above. Cigarette smoking has no influence on the development of mesothelioma. (See N.S. Offermans, et. al., *Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherland Cohort Study,* 56 J. Occupational Envt'l Med. 1 (2014); Robinson BM. *Malignant pleural*

mesothelioma: an epidemiological perspective, 1 Annals Cardiothoracic Surgery 491 (2012)).

Asbestos exposure is the only known occupational and/or environmental cause of mesothelioma in North America, and all of the asbestos varieties induce the genetic errors described above and cause this cancer. The fibers that cause mesothelioma reach the pleural surfaces through the lymphatic pathways, as explained earlier, and they interact with the target cells of the mesothelial surfaces. When a sufficient number of genetic errors have accumulated in a single mesothelial cell, this cell can undergo neoplastic transformation and grow into a deadly tumor. It typically takes many decades for a sufficient number of mutations to occur in a single mesothelial cell because of the numerous effective defense mechanisms that destroy genetically defective cells, thus explaining the long latencies known for this cancer. However, shorter latency periods have been reported in the literature, and latency period is measured as the time from first exposure; all additional exposures to asbestos are cumulative.

All of the asbestos varieties have been shown to cause genetic errors and fibers less than five microns can bind DNA and this contributes to the development of genetic damage. Short fibers have been found to accumulate in the pleural regions of the lung as well as in mesenteric lymph nodes of the peritoneal cavity. Longer fibers may be comparatively more dangerous than short fibers (on a fiber per fiber basis), but all size ranges are capable of causing and contributing to the development of mesothelioma or any of the asbestos-related diseases. Exposure to asbestos fibers of all types and lengths are toxic, and short fibers more readily reach the mesothelial target cells of the pleura. (See Y. Suzuki & S. R. Yeun, *Asbestos Fibers Contributing to the Induction of Human malignant mesothelioma.,* 982 Annals N.Y. Acad. Sci. (2002); Y. Suzuki, et al. *Short thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence.,* 208 Int'l. J. Hygiene Env. Health 201 (2005)). Some have suggested that geological nomenclature – calling the anthophyllite and tremolite in the talc either "non-asbestiform" or "cleavage fragments" – has biological significance. This notion has been rejected by the EPA, US Centers for Disease Control and Prevention, Agency for Toxic Substances and Disease Registry USGS (United States Geological Survey), and American Thoracic Society, and most recently by the FDA Working Group, and is not a distinction that is considered medically important. The Finnish Institute of Occupational Medicine (2019) provided a definition of asbestos that states that "asbestos fibers with a thickness of 3 micrometers or less and a length of 5 micrometers or more cause a risk of cancer and pulmonary diseases when inhaled, regardless of whether they have been formed as a result of a geological process metamorphism or in an industrial process, such as in mining operations." In fact, mesotheliomas have been documented among New York State miners and millers of talc containing approximately 50% "non-asbestiform" anthophyllite and tremolite. Asbestos related diseases have also been found at the Italian and Vermont talc mines and mills. The absence of documented cases of mesothelioma among one cohort of miners and millers of talc containing less than 1% the tremolite and anthophyllite (such as the Rubino, Coggiole, and Pira Italian studies of talc miners and millers) is most likely due to an inadequate sample size, selection criteria, and the manner in which the data has been reported. (US EPA Region 9 Response to the 2005 National Stone, Sand and Gravel

Association Report, April 20, 2006; RT Vanderbilt Co., MSDS, May 1, 1975; Roggli, et.al. *Tremolite and Mesothelioma.* Ann Occ Hyg 46(5):447-453 (2002); Lamm, *Similarities in Lung Cancer and Respiratory Disease Mortality of Vermont and New York State Talc Workers;* Epidemiology-Fibers, 1576-1581 (1988), Mirabelli, *Letter on "Cosmetic Talc as a Risk Factor for Pleural Mesothelioma: a Weight of Evidence Evaluation of the Epidemiology;* Inhal Toxicol 29(8):341 (2017)). Mirabelli noted a case of mesothelioma in a 79 year old man who was a maintenance worker who worked at the Italian talc mill from 1947-1957 (Mirabelli D, Letter on: "Cosmetic talc as a risk factor for pleural mesothelioma: a weight of evidence evaluation of the epidemiology, Inhal Toxicol 29(8):341 (2017). A mesothelioma in Johnson & Johnson's consumer product division worker population has been reported. This individual's exposure in the consumer product division would be more analogous to what a consumer of talcum powder products would experience than a miner or miller's exposure. Johnson & Johnson's own internal records document many dozen mesotheliomas with Johnson & Johnson associating the possible cause to be exposure to its talcum powder products.

Fibers of all lengths can bind to DNA and cause genetic errors that are required in the causation of cancer such as mesothelioma. Fiber burden studies of mesothelioma patients show a preponderance of chrysotile asbestos within the tumor tissue. Since the target location of mesothelioma is the pleura, the lung burden of asbestos does not reflect the fact that asbestos has moved from the lung to the pleura, where it can cause the mesothelioma to develop. (See Ronald F. Dodson, *Analysis and Relevance of Asbestos Burden in Tissue, in Asbestos: Risk Assessment, Epidemiology and Health Effects.* Risk Assessment, Epidemiology and Health Effects 78 (2d, ed. 2011); M. Silverstein, et al., *Developments in Asbestos Cancer Risk Assessment.* Am J. of Indus. Med. (2009)).

Moreover, there is ample evidence to support the conclusion that exposure to the asbestos fibers typically used in brake linings-chrysotile fibers-can and does cause mesothelioma. This conclusion is supported by, among others, the American Conference of Governmental Industrial Hygienists, the American Thoracic Society, the Environmental Protection Agency, the International Agency for Research on Cancer, the National Toxicology Program, OSHA, the Consumer Products Safety Commission, the World Health Organization, and the World Trade Organization. The scientific consensus that all fiber types and sizes can cause mesothelioma is also reflected in the Consensus Report of the 1997 Helsinki Conference (discussed below) and publications from the American Cancer Society and the National Cancer Institute of the National Institutes of Health.

In essence, there exists a consensus among the overwhelming majority of medical and scientific professionals and organizations that asbestos fibers of any type or size can cause mesothelioma, including chrysotile fibers. (See Dodson, Ronald F. et al., *Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review,* 44 Am J. Indus. Med. 291 (2003); D. Egilman, et al., *Exposing the "Myth" of ABC, "Anything But Chrysotile: A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies.* 44 Am J. Indus. Med. 540 (2003); David S. Egilman & Marion Billings: *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability,* 11 Int. J. Occupational Envtl Health 360 (2005). 11:360-371; Egilman D. *Fiber*

Types, Asbestos Potency, and Environmental Causation. 15 Int. J. Occupational Envtl. Health (2009); Finkelstein, M. *Asbestos Fiber Concentrations in the Lungs of Brake Workers: Another Look,* 52 Annals Occupational Hygiene 455 (2008); M.M. Finkelstein & C. Meisenkothen, *Malignant Mesothelioma among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos.* 54 Annals Occupational Hygiene 692 (2010); P.J. Landrigan, et al., *The Hazards of Chrysotile Asbestos, a Critical Review.* 37 Indus. Health 271 (1999); W.J. Nicholson, *The Carcinogenicity of Chrysotile Asbestos-A Review.* 39 Indus. Health 57 (2001); R.A. Lemen, *Chrysotile Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model.* 10 (2) Int. J. Occupational Envtl. Health (2004); *see also* R. Lemen, *Asbestos in Brakes: Exposure and Risk of Disease.* 45 Am. J. Indus. Med 229 (2004); EPA: *Guidance For Preventing Asbestos Disease Among Auto Mechanics.* (1986); A.H. Smith & C.C. Wright, *Chrysotile Asbestos is the Main Cause of Pleural Mesothelioma.* 30 Am. J. Indus. Med. 252 (1996); U.S. Dept. of Labor: *Working Safely with Asbestos in Clutch and Brake Linings.* (posting); U.S. Dept. of Labor, OSHA Directorate of Science, Technology and Medicine, Office of Science and Technology Assessment. *Asbestos-Automotive Brake and Clutch Repair Work*; World Health Organization, *Environmental Health Criteria 203: Chrysotile Asbestos.* International Programme on Chemical Safety (1998 Geneva)).

Asbestos fibers are very small; so small, in fact, that millions of fibers could fill the air in a room without anyone being able to perceive it with the naked eye. The fibers are odorless, cannot be seen with the naked eye, and are aerodynamic. Consequently, someone can inhale asbestos fibers without even being aware of it. The fibers are also small enough to pass through the normal respiratory defense mechanisms that the human body uses to keep out toxins and debris.

The Scientific community has even concluded that small amount of asbestos exposure can cause cancer. The Rodelsperger study indicates that exposure to asbestos below the Occupational Safety and Health Administration (OSHA) Permissible Exposure Level (PEL) of 0.1 fibers per cubic centimeter can cause disease. However, visible asbestos-laden dust that is released into the air from the manipulation of gaskets or packing, or that is reintroduced into the respirable zone from the process of sweeping the floor, is between 2.0 and 10.0 fibers per cubic centimeter. These levels far exceed the OSHA PEL. Some of these levels even exceed the OSHA PEL issued in 1972.

Government agencies and international organizations universally recognize asbestos as a carcinogen in low levels. These agencies include the International Agency for Research on Cancer, Environmental Protection Agency, OSHA, National Institute for Occupational Safety and Health, and World Health Organization. The inhalation of asbestos fibers also does not trigger any immediate physiological reactions: the victim doesn't experience any immediate irritation, asthmatic problems, or allergic reactions. Moreover, the latency, or development period, for mesothelioma is very long: the minimum latency period is usually considered to be around 10 years with a maximal latency period well over 60 years after the last exposure. There are some case reports of shorter latency periods; the idea of latency starts with the first exposure to asbestos, with additional exposures also contributing. Consequently, it could be decades before someone is aware

that he or she was exposed to asbestos, or it might have occurred so remotely that they do not realize they had asbestos exposure. Moreover, they may not realize that a product they used contained asbestos and thus are unaware they had exposure.

The Helsinki Criteria for Attribution: In January 1997, a conference called "Asbestos, Asbestosis and Cancer" was held in Helsinki, Finland. The conference was convened to establish criteria for diagnosis and attribution of disorders of the lungs and pleura, including mesothelioma. This was a multidisciplinary group of internationally recognized experts, consisting of pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. Collectively, the members had published over 1,000 articles on asbestos and associated disorders. The conclusions of the conference were developed into a peer-reviewed Consensus Report that established the "Helsinki Criterion". Among the conclusions of the Helsinki Criterion are:

a. That, in general, reliable work histories provide the most practical and useful measures of occupational asbestos exposure; and
b. That even in the absence of other independent evidence of disease (e.g. lung fiber counts exceeding the background range for the lab in question; the presence of radiographic or pathological evidence of asbestos-related tissue injury; histopathologic evidence of abnormal asbestos content), a history of significant occupational, domestic or environmental exposure to asbestos will suffice for attribution of the disease with asbestos exposure.

Moreover, with reference to determining an occupational etiology of mesothelioma, the Helsinki Criterion Consensus Report concluded that:

a. The great majority of mesotheliomas are due to asbestos exposure;
b. Mesothelioma can occur in cases with low asbestos exposures. However, very low background environmental exposures carry only an extremely low risk;
c. About 80% of mesothelioma patients have had some sort of occupational exposure to asbestos (necessitating a carefully obtained and detailed occupational history for proper diagnosis);
d. An occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related;
e. A minimum of 10 years from the first exposure is required to attribute mesothelioma to asbestos exposure (though in most cases, the latency interval is longer);
f. Smoking has no influence on the risk of mesothelioma.

The conclusions of the Helsinki Criterion have since been adopted by, and form the general consensus of, the medical community's positions vis-à-vis mesothelioma and asbestos. (See *Consensus Report, Asbestos, asbestosis and cancer: the Helsinki criteria for diagnosis and attribution,* 23 Scandinavian J. Work Environ Health 311 (1997)). And, given the fact that about 80% of patients with mesothelioma have had some sort of

occupational exposure to asbestos,[1] asbestos exposure in the workplace is a prime focus of Occupational Medicine when dealing with mesothelioma patients.

Mesothelioma is a dose responsive disease: It is my opinion that Mesothelioma and asbestos related lung cancer are dose responsive diseases in which more substantial exposures directly increases the risk for the development of these cancers. This linear dose-response relationship presented in *Asbestiform Fibers: Non-occupational Health Risks,* published by the National Research Council National Academy of Sciences in 1984, discussed herein, is neither new nor novel and generally accepted in the medical and scientific communities. As per the aforementioned Helsinki criteria, the first question usually asked of a patient diagnosed with mesothelioma, concerns how, when, and where the patient was exposed to asbestos. (*See Consensus Report, Asbestos asbestosis and cancer: The Helsinki criteria for diagnosis and attribution.* 23 Scandinavian J. Work Environ Health 311 (1997)). Because of the proven association between asbestos fibers and mesothelioma, proof of significant exposure to asbestos dust is considered to be proof of specific causation. (*See* P. Boffetta, et al., *Health Effects of Asbestos Exposure in Humans: A Quantitative Assessment.* 89 (6) Medicina Del Lavoro, 471 (1998). This causal relationship between exposure to asbestos dust and the development of mesothelioma is so firmly established in the scientific literature that it is accepted as a scientific "fact".

Malignant mesothelioma is, in general, a dose response disease where all significant exposure to asbestos-containing dust has been shown to contribute to cause diffuse malignant mesothelioma including pleural mesothelioma (*See also* Newman, et al., *Malignant Mesothelioma Register 1987-1999.* 74 Int'l Arch Env. Health 383 (2001), (concluding that "higher cumulative asbestos-fiber dose leads to the earlier development of mesothelioma)). As each exposure to asbestos contributes to the total amount of asbestos that is inhaled, and, in doing so, reduces the necessary period for asbestos disease to develop. As a result, all non-trivial exposure to asbestos should be considered a contributing factor in the development of the malignant mesothelioma or lung cancer. More recently, the BAP-1 gene mutation has been found to confer increased susceptibility in individuals who have both the mutation and have asbestos exposure. There is no evidence that the genetic mutation of this tumor suppressor gene without asbestos exposure causes mesothelioma.

Exposure to Asbestos contaminated talc and disease: Asbestos fibers have been reported in cosmetic talcum powder for decades, in company documents, the media, FDA communications, and the published medical and scientific literature. In 1935 asbestos was identified as a source of exposure in talc miners and millers by Dreesen. By 1968 Cralley had described asbestos in consumer cosmetic talc products. By 1972, the cosmetic industry was looking for asbestos free alternatives to cosmetic talc. Cosmetic talc has been analyzed by researchers in various countries, and has routinely been shown to be contaminated with asbestos. In 1957, researchers at Battelle Memorial reported finding tremolite in Italian talc used by Johnson & Johnson. In 1968, Johns-Manville documented fibrous tremolite asbestos in consumer cosmetic talcum powder products. In 1972, Snider, et al., reported

[1] The remaining 20% of mesothelioma patients likely had asbestos exposures that were para-occupational or are simply unidentified.

finding asbestos in several consumer cosmetic talcum powder products, including Johnson's Baby Powder. That same year, Lewin of New York University reported finding asbestos in numerous talcum powder products including Johnson's Baby Powder purchased off the shelf. The University of Minnesota also found asbestos in Lewin's sample of Johnson's Baby Powder. In 1973, Lewin reported asbestos in Clubman talc. That same year, talc industry Round Robin testing reported asbestos in Italian, Montana, Alabama, and North Carolina talc sources. In 1974 Rohl, and in 1976, Rohl and Langer tested 20 consumer products that had been labeled as talc or talcum powder, including body powders. Of the 20 products that were tested, ten were found to contain tremolite and anthophyllite, principally asbestiform. Of note, the product that had the highest asbestos content in the Rohl and Langer study was the same product later tested by Gordon, et.al. Pooley, while consulting for Johnson & Johnson, also found anthophyllite in the Cashmere Bouquet Rohl and Langer studied. In 1979, Berg reported the presence of asbestos in the Montana talc mine deposits, the source of talc used to manufacture Clubman and included in Mennen talc. In 1991, Blount reported amphibole asbestos in talc from Vermont used to manufacture Johnson's Baby Powder. Mattenklott et. al. in 2007 found that low or trace levels of asbestos by weight in talcum powder (0.1 percent) released millions of asbestos fibers upon use. In 2015, Ilgren, et al. attributed the increased rate of mesothelioma in the chrysotile miners in Italy to the tremolite asbestos in the talc in the adjacent mining region. Recently, Saldivar under contract for FDA reported chrysotile asbestos in Johnson's Baby Powder.

Exposure to asbestos-containing talc has been shown to cause asbestos related diseases, including mesothelioma. A paper by Gordon, et.al., Asbestos in Commercial Cosmetic Talcum Powder as a Cause of Mesothelioma in Women, evaluated the mineralogical constituents of Cashmere Bouquet and its ability to release asbestos fibers into the breathing zone of the direct user and bystanders. In their paper Gordon et.al. noted that the talc that was used in Cashmere Bouquet was derived from three distinct regions, where anthophyllite and tremolite asbestos were found, regions from which the talc used in Johnson's Baby Powder was also sourced. Gordon et.al. measured 18 million anthophyllite asbestos fibers per gram in the talcum powder. Air measurements were done by both phase contrast microscopy (PCM) and transmission electron microscopy (TEM), and significant levels of asbestos fibers were noted (anthophyllite, tremolite and some chrysotile) in the breathing zone of the individual applying the powder as well as a bystander. Results taken from the experiment in the paper show that personal measurements from the shaker container test showed a measurement by PCM of 4.8 f/cc, with an actual asbestos fiber measurement of 1.8 f/cc. Bystander measurements showed a lower, but still significant exposure of 1.35 f/cc by PCM for the bystander, and 0.5 f/cc of actual asbestos fibers. Similar measurements were done with the puff application method. Personal measurements after using a puff were 23.6 f/cc and 16.5 f/cc for the user, with actual asbestos fiber measurements of 5 f/cc and 3.5 f/cc. A short term sample showed even higher measurements, of 60 f/cc with the use of a puff and actual asbestos fiber measurements of 13 f/cc. Bystander exposures to asbestos from the puff application were elevated, with a short term sample by PCM of 13.7 f/cc and 9.7 f/cc, and an actual asbestos fiber measurement of 4.9 f/cc and 3.5 f/cc. Gordon et.al. also noted that the TEM measurements were far more sensitive than x-ray diffraction detection, since there was a

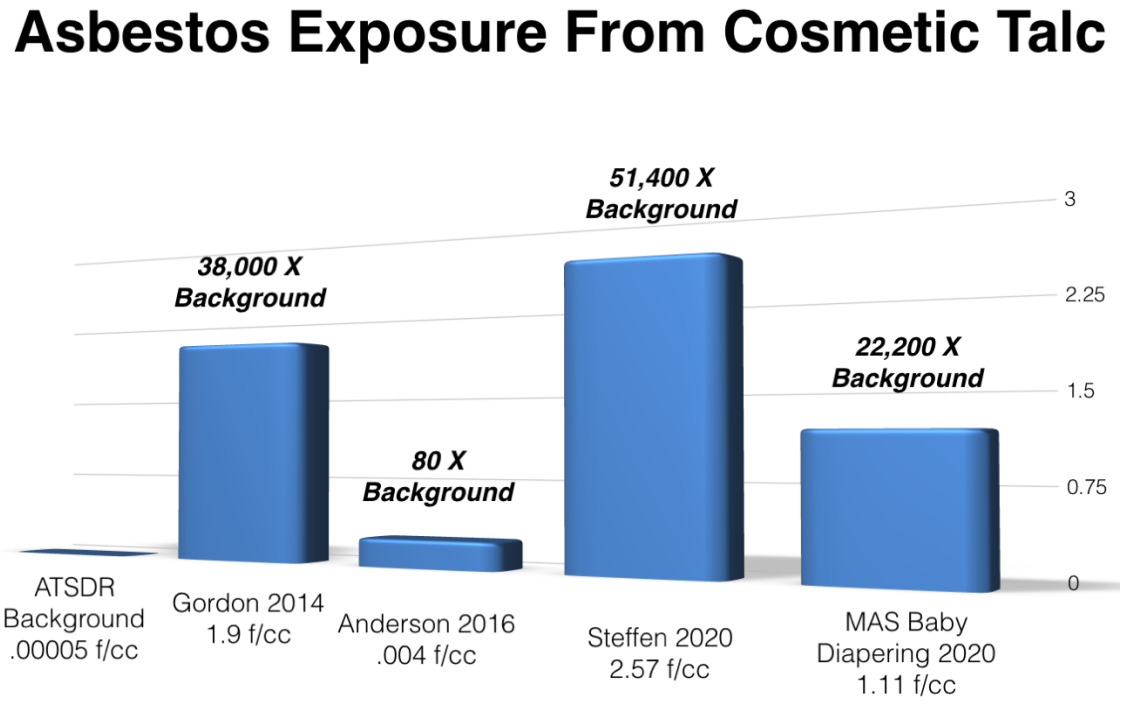
much lower detection limit with TEM. In addition, the Mine Safety and Health Administration (MSHA) monitored personnel in the mill where Italian talc was ground (this talc was used in consumer products) in 1984. The filters form the personal measurements from these workers contained 5.8% anthophyllite. The MSHA scientist determined that this equated to anthophyllite comprising 0.6% of the bulk Italian talc. Dr. Steven Compton found asbestos (anthophyllite, tremolite, actinolite, and chrysotile) in 11 samples collected from the Italian mining region from which the talc originated that was then used in consumer products, including Cashmere Bouquet and Johnson's Baby Powder and others. Dr. Compton further found asbestos in 6 of 7 Argonaut Vermont talc mine samples and 4 additional samples collected on the mining property, as well as in samples of Montana talc provided to him by the manufacturer of Clubman.

Both historic and recent analyses (published in the medical and scientific literature as well as industry, government and private laboratory testing) of the talc from the source mines used in Johnson's Baby Powder and Clubman finished powder products, have shown significant amounts of chrysotile, anthophyllite and tremolite asbestos. Studies done recently from products using ore taken from the same source mines as those used in the manufacture of the Johnson's Baby Powder and Clubman, showed significant amounts of chrysotile, anthophyllite, and tremolite asbestos. The asbestos found in the ores is also found in the finished consumer products. Dr. William Longo from MAS found tremolite and anthophyllite, as well as chrysotile, in his analysis of historic and current Johnson's Baby Powder and Shower to Shower powder products, the same fiber types found in the historic testing records of Italian and Vermont talc ores and in historic testing records of Johnson's Baby Powder. Dr. Longo analyzed and reported on approximately 146 Johnson's Baby Powder and Shower to Shower products that were manufactured from the 1940s through the 2000s and provided to his laboratory from mesothelioma plaintiffs, off-the-shelf purchases, from collectors, and from Johnson & Johnson's own historic collection (72 obtained directly from Johnson & Johnson). Dr. Longo report regulated asbestos in 116 of the 146 (79%) containers of Johnson & Johnson talcum powder products. For Johnson & Johnson talcum powder products manufactured in the United States, Dr. Longo reported regulated asbestos in 88 of 112 containers (79%). For containers manufactured in the United States after 2003, Dr. Longo report asbestos in 37 of 40 (93%) of containers. Subsequent analyses have shown chrysotile present in Johnson's Baby Powder sourced from Chinese talc as well. MAS and MVA (Dr. Compton's laboratory) have analyzed and identified asbestos in hundreds of containers of talcum powder products and ore samples from varying vintages and sources.

Dr. Longo's laboratory has performed additional analysis to determine whether exposure from use of talcum powder below the waist (using Italian talc-sourced Johnson's Baby Powder), led to respirable levels of asbestos. Samples taken in the breathing zone of an individual during use of Johnson's Baby Powder below the waist, resulted in a mean tremolite fiber exposure of 2.57 fibers/cc. Area samples taken resulted in a mean tremolite fiber exposure of 0.2 fibers/cc. Dr. Longo's results were published in 2020 (Steffen, 2020). The cosmetic talc testing done by Dr. Longo and Dr. Compton, combined with published air measurements in the medical literature (Gordon 2014, Anderson 2016, Steffen 2020) all show measurements of asbestos that are orders of magnitude above the ATSDR

background level of 0.00005 f/cc. At the levels measured by these authors, the literature shows a substantially increased risk for disease at the cumulative levels of exposure (Iwatsubo, Rodelsberger, LaCourt).

Exposure data from talcum powder usage as compared to background is identified on the graph below:



In addition to looking at bulk and air samples, Gordon et.al analyzed the lung tissue and lymph node tissue of a woman who had been exposed to contaminated talcum powder (Cashmere Bouquet). The authors found that there were 3150 and 4150 fibers per gram wet weight, respectively, with a detection limit of 690 fibers per gram wet weight. All fibers were 5 micrometers or greater in length, and had an aspect ratio of 20:1 or greater. The fibers were identified as anthophyllite or tremolite. In addition to the fibers counted above, there were many anthophyllite and tremolite fibers that were less than 5 micrometers in length, with a predominance of anthophyllite. In the lymph node, amphibole asbestos fibers were also noted, measuring 12,738 fibers per gram wet weight (detection limit 2123 fibers per gram wet weight). Again, the fibers noted were anthophyllite and tremolite. In addition to the asbestos found in the lungs, the authors noted fibrous and platy talc and small asbestos bodies.

The issue of asbestos and talc has been studied for decades. Millman in 1947 noted pneumoconiosis in a man exposed to cosmetic talc. Lung scarring was seen in miners from New York State in the 1950s, and there are elevated rates of mesothelioma and lung cancer in miners at the asbestos contaminated talc mines. Moskowitz 1970 reported a talc pneumoconiosis in a woman exposed to cosmetic talc while working as a quality control inspector on the production floor at Revlon for 11 years. The International Agency for Research on Cancer has noted that talc contaminated with asbestos is carcinogenic. Case

reports published out of Italy have involved patients with mesothelioma and exposure only to cosmetic talcum powder (Andrione, 1994, Musti, 2009). In the fall of 2019, the FDA found asbestos contamination in Johnson's Baby Powder, leading to a recall of thousands of bottles of cosmetic talc. In 2018, OSHA found tremolite asbestos contamination in make-up products that contained cosmetic talc. I have recently published a paper, along with co-authors, that describes 33 cases of mesothelioma among individuals whose only known exposure to asbestos was through their use of cosmetic talc (Moline et al, 2019). Emory et al. (2020) has published a paper on an additional 75 individuals with mesothelioma whose source of asbestos exposure was cosmetic talc. Together, these papers show over 110 patients with mesothelioma and cosmetic talc use.

Applying an Accepted Method for Evaluating Disease Causation in an Individual:

In deciding whether Mr. Gref's mesothelioma was caused by his exposure to asbestos, I applied the methodology that was described by Welch, et.al. in her paper Asbestos Exposure Causes Mesothelioma, but Not This Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court, published in 2007 in the International Journal of Occupational and Environmental Health. This method mirrors the Hill criteria, but is specific for asbestos (see also Lemen). Similar methodology for assessing causation for individuals exposed to asbestos who developed asbestos-related diseases was also outlined by Freeman. In this paper, Dr. Welch identifies four questions that should be examined in the causation of disease in an individual:

1. Was the individual exposed to a toxic agent?
2. Does the agent cause the disease present in the individual?
3. Was the individual exposed to this substance at a level where the disease has occurred in other settings?
4. Have other competing explanations for the disease been excluded?

For question #2, there is ample literature that asbestos causes mesothelioma and no dispute in the medical literature. With respect to question #1, Mr. Gref was exposed to asbestos from talcum powder for approximately 36 years, from birth continuing until around 2018, fulfilling this criterion. Clubman talcum powder, Mennen, Johnson's Baby Powder, Old Spice, and other talcum powders using talc from the same ore sources have been shown to contain asbestos, and Mr. Gref would have had asbestos exposure based on his family's descriptions of their use on him as well as his own descriptions of his powder use. For criteria #4, Mr. Gref had no known alternate exposures to asbestos. The remaining criterion, #3 is whether there is an analogous exposure scenario in which others also developed mesothelioma. As described above, and recently referenced by the Center for Disease Control, as well as published in the peer-reviewed literature, there are numerous other individuals with exposure to asbestos-containing talc products who have developed malignant mesothelioma.[2]

[2] Andrion, Alberto, et al. *Malignant Peritoneal Mesothelioma in a 17-Year-Old Boy with Evidence of Previous Exposure to Chrysotile and Tremolite Asbestos*, Human Pathology, Volume 25, No. 6 (June 1994); Bulbulyan, M.A., et al., *Cancer Mortality Among Women in the Russian Printing Industry*, AM J Ind Med, 36:166-171(1999); Finkelstein, M., *Malignant Mesothelioma Incidence Among Talc Miners and Millers in New York State*, Am J Indust Med 55, 863-868 (2012); Ghio, A, Roggli, V, *Talc Should Not Be Used for Pleurodesis in Patients with Nonmalignant Pleural Effusions*, Am J Respir Crit Care Med, Vol 164, No. 9,

Summary and Specific Causation in Mr. Gref's Case

Based on the information that was provided to me and the diagnosis of mesothelioma as outlined by his treating physicians, and applying both my understanding of the medical and scientific literature and the facts of this case, it is my opinion to a reasonable degree of medical certainty that the exposures to the dust from asbestos-contaminated cosmetic talc products that Mr. Gref used or were used on him, beginning approximately 36 years prior to his diagnosis, were above normal background levels. Both historic and recent analyses (published in the medical and scientific literature as well as industry, government and private laboratory testing) of the talc from the source mines used in Johnson's and Johnsons' Baby Powder, Old Spice, Mennen, Clubman talc have shown significant amounts of asbestos, including chrysotile, anthophyllite and tremolite asbestos.

Studies done recently on both Clubman talc products and on products using ore taken from the same source mines as those used in the manufacture of Clubman Talc showed significant amounts of chrysotile, anthophyllite, and tremolite asbestos. Dr. Compton found asbestos (primarily anthophyllite) in 6 different containers of Clubman talc manufactured with Montana talc. Recent testing of two Clubman samples showed tremolite/actinolite asbestos, ranging from 11.8 million fibers per gram in one container to 2.3 million fibers/gram in the other (although the latter measure is likely an underestimate due to a higher concentration of fibers initially noted on direct examination). Dr. Compton also identified asbestos in a Clubman container from a vintage prior to the use of Montana talc. Dr. Compton analyzed and identified asbestos in samples of Montana talc produced by the manufacturer of Clubman.

Studies by Dr. Longo have also identified asbestos in an Avon Night Magic container manufactured with Montana talc. While Mr. Gref did not use Avon products, the talc used came from the same region. Similarly, both Drs. Longo and Compton have identified asbestos in Cashmere Bouquet manufactured with Italian and Montana talc. These findings are consistent with both current and historic product testing of talcum powder products containing the same talcs (Cashmere Bouquet – 5/5 Montana talc from Beaverhead; Avon Night Magic – Montana; Clubman – 6/6 Montana talc from Barretts Minerals and Whittaker Clark & Daniels). Testing of Old Spice powders by Dr. Longo has shown asbestos in virtually every container evaluated as well. Similarly, Dr. Mark Kreleker has done a comprehensive review of testing of the ore and talcum samples over decades,

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pp 1741 (2001); Fujiwara, Hiroshi, et al. *An Autopsy Case of Primary Pericardial Mesothelioma in Arc Cutter Exposed to Asbestos through Talc Pencils*, 43 Industrial Health 346-350 (2005); Ilgren E, et al., *Critical reappraisal of Balangero chrysotile and mesothelioma risk*, Epidemiology Biostatistics and Public Health, Vol. 12, No. 1 (2015); Lamm, S.H., et al., *Similarities in Lung Cancer and Respiratory Disease Mortality of Vermont and New York State Talc Workers*, Epidemiology-Fibers, 1576-1581, (1988); Mirabelli D, *Letter on: "Cosmetic talc as a risk factor for pleural mesothelioma: a weight of evidence evaluation of the epidemiology"*, Inhalation Toxicology, 29:8, 341 (2017); Musti, et al., *Exposure to Asbestos and Mesothelioma Risk of Onset of Primary Ovarian, Description of Two Cases*, (2009); Moline, Jacqueline, et al., *Mesothelioma Associated with the Use of Cosmetic Talc*, Journal of Occupational and Environmental Medicine, (2020); Emory, Theresa, et al., *Malignant mesothelioma following repeated exposures to cosmetic talc: A case series of 75 patients*, Am J Ind Med, (2020).

documenting testing results showing that asbestos was repeatedly found, including in the Italian, Montana, Chinese and North Carolina sources.

Fiber release studies done recently by MVA Scientific Consultants and others from products using ore taken from the same source mines as those used in the manufacture of Johnson's Baby Powder and Mennen (which used the same talc as that used in Cashmere Bouquet) showed significant amounts of chrysotile, anthophyllite, and tremolite asbestos. MSHA found anthophyllite in the mills that processed the Italian talc. Similarly, Dr. Compton found anthophyllite in 11 of the 13 samples of talc ore from the Italian mines, from which the talc originated that, was then used in consumer products. As outlined above, MAS performed an additional analysis to determine whether exposure of talcum powder below the waist led to respirable levels of asbestos (using Johnson and Johnson Baby Powder). Their report stated that there was a mean tremolite fiber exposure of 2.57 fibers/cc during this activity. Dr. Longo has done extensive testing on Chinese talc, which was used in both Johnson's Baby Powder, and has found chrysotile asbestos in every sample he has evaluated. Dr. Longo has evaluated 91 containers of Cashmere Bouquet, and found asbestos in 79 of 91 containers.

Alternative powders not containing talc were available since the early 20th century. The opinions related to Mr. Gref's case are based on my review of the evidence of exposure in this case, the medical and scientific literature as described above regarding asbestos exposure and disease, available studies concerning fiber release, epidemiological studies of exposure to asbestos exposure and the development of disease, and my knowledge, skill, experience, and training as a physician specializing in occupational medicine with a clinical focus on evaluating individuals with asbestos exposure.

In conclusion, Mr. Gref's exposure to asbestos-contaminated talcum powder led to his diagnosis of peritoneal mesothelioma. He has undergone chemotherapy and cytoreductive surgery. There is no cure for mesothelioma, and his prognosis is poor.

Sincerely,

Jacqueline Moline, MD, MSc, FACP, FACOEM